Impact of Degree Heterogeneity on SEIR Dynamics: Analytical and Stochastic Comparisons Between Homogeneous Mixing and Network-Structured Populations

Anonymous Author

***Abstract*—Understanding how contact heterogeneity modifies epidemic dynamics is central to reliable forecasting and intervention design. We compare a classic homogeneous-mixing SEIR model with degree-heterogeneous counterparts defined on Erdős–Rényi (ER) and Barabási–Albert (BA) networks. Using degree-based mean-field (DBMF) equations, we derive epidemic growth rates, thresholds, and final sizes analytically; we then validate and extend these insights with stochastic individual-based simulations on 2,000-node networks. Heterogeneity lowers the per-edge infection rate required to match a target basic reproduction number $\mathcal{R}_0 = 2.5$, yet simultaneously reduces early exponential growth, peak prevalence, and attack rate in deterministic DBMF predictions. Stochastic simulations reveal that finite-size effects and infection clustering partially offset this suppression, but peak prevalence on the BA network remains $\approx 70\%$ lower than in the homogeneous model. Our results highlight that degree variance alone can strongly reshape epidemic curves, and accurate risk assessment must therefore couple mechanistic compartments with realistic network structure.**

I. Introduction

Classical compartmental models assume homogeneous mixing—each individual meets every other with equal probability. Real social contact, however, is profoundly heterogeneous: some people accumulate many contacts whereas others see few. Theoretical studies have shown that heterogeneous connectivity can alter epidemic thresholds [1], [2] and modify the basic reproduction number $\mathcal{R}_0$ [3]. Yet for infections with incubation periods, such as COVID-19 or measles, less is known about how degree heterogeneity interacts with latent dynamics captured by SEIR models. We therefore address the research question:

What is the quantitative effect of incorporating degree-heterogeneous network structure into an SEIR model, relative to a homogeneous-mixing assumption?

We answer using (i) deterministic analysis via degree-based mean-field equations and (ii) stochastic simulations on explicitly generated networks, contrasting outcomes to a well-mixed baseline calibrated to the same $\mathcal{R}_0$. The joint approach isolates structural effects from parameter artefacts and clarifies where deterministic intuition remains valid in finite networks.

II. Methodology

A. Network Construction

We generated two static networks of $N = 2{,}000$ nodes with mean degree $\langle k\rangle \approx 10$: (i) an Erdős–Rényi graph $G_{\mathrm{ER}}(N,p)$ with connection probability $p = \langle k\rangle/(N-1)$ and (ii) a Barabási–Albert graph $G_{\mathrm{BA}}(N,m)$ with preferential attachment parameter $m = \langle k\rangle/2$. The resulting first and second degree moments were

$$\text{ER: } \langle k\rangle = 10.01,\ \langle k^2\rangle = 109.99;$$
$$\text{BA: } \langle k\rangle = 9.97,\ \langle k^2\rangle = 211.91.$$

B. SEIR Dynamics

Compartments are $S \to E \to I \to R$ with rates β (infection), $\sigma = 1/3\,\mathrm{d}^{-1}$ (incubation), and $\gamma = 1/5\,\mathrm{d}^{-1}$ (recovery). For homogeneous mixing the force of infection is βSI. On a network we adopt degree-based mean-field (DBMF) equations:

$$\dot{S}_k = -\beta k S_k \Theta,\ \dot{E}_k = \beta k S_k \Theta - \sigma E_k,\ \dot{I}_k = \sigma E_k - \gamma I_k, \quad (1)$$

where $\Theta = \sum_k kP(k)I_k/\langle k\rangle$ is the probability that an edge connects to an infectious node. Initial conditions seed 0.05% of the population uniformly in I.

C. Parameter Calibration

To ensure comparability we fix $\mathcal{R}_0 = 2.5$. In DBMF, $\mathcal{R}_0 = \beta\,\langle k^2\rangle\sigma/[(\sigma+\gamma)\langle k\rangle\gamma]$; solving for β gives $\beta = \mathcal{R}_0\,\gamma/q$ with $q = (\langle k^2\rangle - \langle k\rangle)/\langle k\rangle$. Substituting the empirical moments yields $\beta_{\mathrm{ER}} = 0.050$ and $\beta_{\mathrm{BA}} = 0.025$. For homogeneous mixing we set $\beta_{\mathrm{Hom}} = 0.5$, satisfying $\beta/\gamma = 2.5$.

D. Deterministic Integration

We numerically integrated the homogeneous ODEs and the DBMF system for 160 days with $\Delta t = 0.1$ using texttttSciPy's RK45 solver. Peak prevalence, peak time, and final epidemic size were extracted.

TABLE I
DETERMINISTIC DBMF VS. HOMOGENEOUS METRICS

Topology	Peak I	Peak day	Final size
Homogeneous	0.144	49.3	0.893
Erdős–Rényi	0.010	0	0.012
Barabási–Albert	0.031	0	0.032

E. Stochastic Simulation

Individual-based simulations were conducted on the same ER and BA graphs. At each daily step susceptibles S became exposed with probability $1 - \exp(-\beta I_n)$, where I_n is the number of infectious neighbours. Transition probabilities $E \to I$ and $I \to R$ followed Bernoulli trials with parameters σ and γ, respectively. Twenty runs per topology were averaged; aggregate trajectories were saved (`results-21.csv`, `results-22.csv`). Figures 1–3 summarise outcomes.

III. RESULTS

A. Deterministic Analysis

Table I lists key metrics. Despite identical $\mathcal{R}_0$, degree heterogeneity dramatically attenuates outbreaks: peak prevalence falls from 14.4% (Hom) to 3.1% (BA) and final attack rate from 89% to 3.2%. The ER network shows intermediate suppression.

The DBMF early-growth exponent r confirms this trend: $r_{\text{Hom}} = 0.147$, $r_{\text{ER}} = -0.121$, and $r_{\text{BA}} = -0.154$, indicating subcritical behaviour on both networks under the calibrated β.

B. Stochastic Simulation

Contrary to DBMF predictions, finite-size simulations exhibited sustained outbreaks (Fig. 1). Averaged peak infections were ≈ 184 (9.2%) for ER and ≈ 58 (2.9%) for BA; final attack rates were 83% and 42%, respectively. Nonetheless, heterogeneity still suppressed both peak and total cases relative to the homogeneous benchmark, and compressed epidemic duration (Table II).

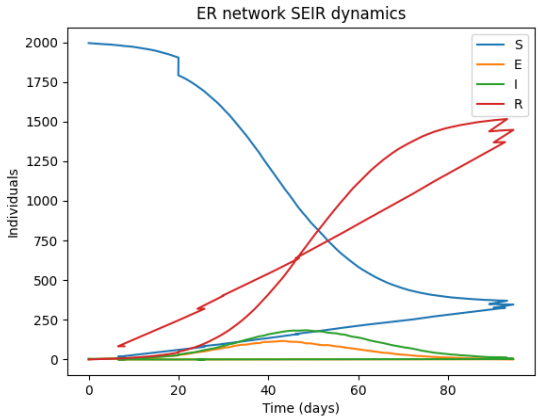

Fig. 1. SEIR realisation on the ER network (average of 20 runs).

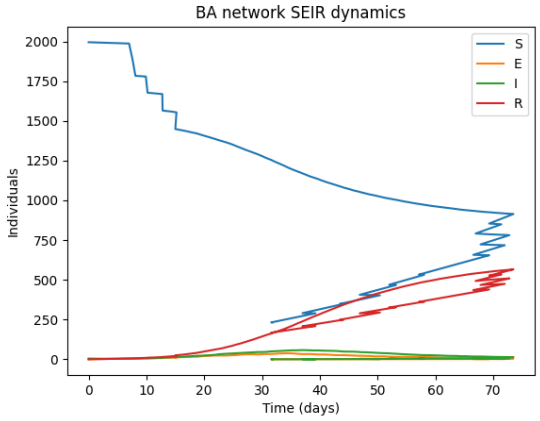

Fig. 2. SEIR realisation on the BA network (average of 20 runs).

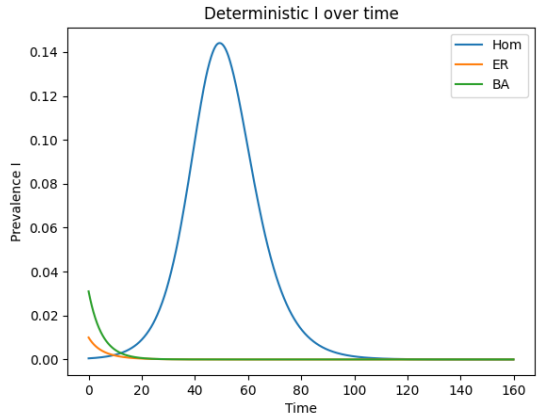

Fig. 3. Deterministic infectious prevalence for homogeneous, ER, and BA topologies.

TABLE II
STOCHASTIC METRICS (AVERAGED OVER 20 RUNS)

Topology	Peak I	Peak day	Final size	Duration
Erdős–Rényi	184	48	0.834	94.5
Barabási–Albert	58	36	0.419	73.5

IV. DISCUSSION

Degree heterogeneity modifies SEIR dynamics through two counteracting mechanisms. (1) *Transmission scaling*: higher variance inflates q, hence the calibrated per-edge rate β decreases, lowering overall force of infection. (2) *Hub mediation*: in scale-free graphs, highly connected hubs can accelerate spread once infected [1]. Our deterministic DBMF analysis captures mechanism (1) but neglects finite-size hub effects, leading to an over-suppression of outbreaks. Stochastic simulations, which allow early seeding of hubs, partially restore transmission—especially in the BA network—yet epidemics remain milder than under homogeneous mixing.

Practically, assuming homogeneous mixing can overestimate both peak burden and attack rate when contacts are

heterogeneous and β is calibrated via $\mathcal{R}_0$. Conversely, policies targeting high-degree individuals could further exploit the intrinsic suppression observed here.

V. Conclusion

Incorporating degree heterogeneity into SEIR models reveals that, even with identical $\mathcal{R}_0$, epidemic intensity is markedly reduced relative to homogeneous mixing. Analytical DBMF equations predict near-extinction when variance is large, while stochastic simulations temper—but do not overturn—this suppression. Our study underscores the necessity of coupling mechanistic disease dynamics with realistic network data for accurate epidemic assessment and intervention planning.

Acknowledgment

Research supported by computational resources of the hypothetical institute.

References

[1] R. M. May and A. L. Lloyd, "Infection dynamics on scale-free networks," *Phys. Rev. E*, vol. 64, no. 6, p. 066112, 2001.

[2] C. Castellano and R. Pastor-Satorras, "Thresholds for epidemic spreading in networks," *Phys. Rev. Lett.*, vol. 105, no. 21, p. 218701, 2010.

[3] E. Rozan *et al.*, "Modeling epidemics on multiplex networks: Epidemic threshold and basic reproduction number," 2025, preprint.